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# A REVIEW OF MOTION SICKNESS WITH SPECIAL REFERENCE TC SIMULATOR SICKNESS

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The incidence of simulator sickness is increasing. As many incidents have been reported since 1980 as in all of time before then, and there are implications for training and safety. The present paper reviews: the signs and symptoms, stimuli and response characteristics, anatomical structures, and susceptibility factors. The prevalent theories of the genesis of this malady are put forth and an integrating theory is proposed which suggests that simulator sickness is a form of motion sickness and may be best understood as a special case of sensory rearrangement. Other works in this series (1) describe the scope of the problem, (2) report the incidence in over 4500 exposures, and (3) list more than 2000 relevant references.

The theory which is offered suggests that motion sickness is a result of decorrelation between and within sensory input channels of information. This correlation theory

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which is offered suggests that motion sickness is a result of decorrelation between and within sensory input channels of information. This correlation theory is in general agreement with the perceptual conflict or cue mismatch theories, and implies that the central nervous system (CNS) perceives decorrelated stimulations as toxic events. This CNS interpretation of toxicity causes the constellation of symptoms associated with motion sickness to be released. Decorrelation can occur when inputs are not in accord with what is expected from past experience or the way that the sensory system may be considered to be "hard-wired" to respond. It is offered that each sensory modality has channels and peak sensitivities and the conflict occurs when spatial (gain) and temporal (phase) aspects of the stimulus are not in accord with each other or with past experience. If this lack of accord occurs with energy to which the two channels are both sensitive, there is more disruption than with sensory inputs where one or the other sensory channel may be insensitive. Sensory conflict is a useful coherent principle in the study of motion sickness because the malady is clearly polygenic and polysymptomatic. Therefore, it may be argued that greater conflict leads to more severe and greater incidence of sickness. From this view, evaluations may be planned which will lead to recommendations for preventing the problem, guidelines for predicting the outcome, and suggestions for future research.

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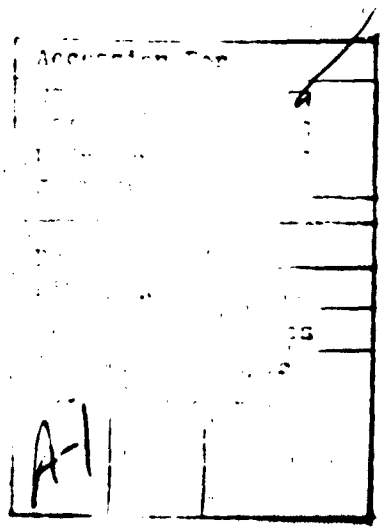

# PREFACE

"Simulator sickness" is not a new phenomenon for those who have worked around training devices, and optical devices which have dynamic characteristics; moreover, to those who have experimented with perceptual adaptation in employing optical transformations, it is not surprising that sickness occurs. For those who perform experiments in vestibular science, simulator sickness may be considered the visual analogue of more traditional forms of motion sickness, including space sickness.

Simulator sickness, while not necessarily of epidemic proportions, is on the upswing. Almost as many incidents have been reported since 1980 as in all of time before then. A sourcebook (Kennedy, Frank, & McCauley, 1984) reports many of these relationships, and a workshop has been held to discuss its implications (McCauley, 1984). Simulator usage is also increasing, and it is problematic as to whether the increase in observed frequency is due to the increased availability of simulators, increased kinematics available in simulated scenarios, or even individual differences of today's user population.

There have been five major (and many minor) reviews of the motion sickness literature (Reason & Brand, 1975; Money, 1970; Chinn & Smith, 1955; Tyler & Bard, 1949; McNally & Stuart, 1942), and all of these contain excellent accounts of what is known. Each has a comprehensive reference list. Additionally, a content-oriented reference list (Kennedy, McCauley, & Miller, 1984) has been assembled which, in our judgment, contains most of the literature which has special relevance for simulator sickness. The following review has been prepared in order to emphasize our bias that simulator sickness may be best understood in the context of the relationship of motion sickness as a special case of sensory rearrangement. In addition, we would like to suggest that this connection (transformed perceptions) may provide heuristic value for the study and understanding of the space adaptation syndrome (Homick, 1979a, b, 1982; Nicogossian & Parker, 1982).

It was our intention here to summarize what we know about motion sickness in order to provide a common baseline of knowledge for the members of the simulator sickness workshop (McCauley, 1984). The phenomenon of motion sickness and the various theories are also briefly presented.

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## INTRODUCTION

While the self-propelled locomotor behavior of man (walking, running, jumping) does not induce motion sickness, transportation in some environments does. It is probably reasonable to assume that the history of motion sickness began with man's domestication of animals for transport. Riding camels or elephants, for example, can induce motion sickness; but interestingly, riding horses does not (Money, 1970). With the invention of the boat and the concomitant increased likelihood of seasickness, motion sickness became more than a problem of discomfort or disquietude. It became a factor that distracted or even prevented a seaman from performing his duties. In short, motion sickness became operationally significant. Thus, the search for the causal factor(s) of motion sickness probably received its initial impetus from a practical concern about how to eliminate the debilitating effects.

This practical aspect of motion sickness is still of significance today. Since the invention of the boat, the variety, speed, and maneuverability of real and simulated transport vehicles have greatly increased. Many of these nonphysiologic environments (e.g., aircraft, automobiles, spacecraft, and vehicle simulators) can cause motion sickness. But why do some vehicles induce motion sickness, while others, such as motorcycles, do not? Why are some people more susceptible to motion sickness than others? What is motion sickness? Is there a theory that explains and helps us understand the phenomenon? Is there a commonality among motion sickness, simulator sickness, and space sickness?

# SECTION I

## MOTION SICKNESS - A DEFINITION

Motion sickness is a general term for a constellation of symptoms and signs, generally adverse, due to exposure to abrupt, periodic, or unnatural accelerations. One must have organs of equilibrium for the malady to develop (James, 1882; Fregly & Kennedy, 1965; Kennedy, Graybiel, McDonough & Beckwith, 1968). Overt manifestations (signs) are pallor, sweating, salivation, and vomiting (Kennedy & Graybiel, 1963a, b; Wiker, Kennedy, McCauley & Pepper, 1979a, b). Drowsiness, dizziness and nausea are the chief symptoms. Less frequently reported, but often present, are postural changes, or ataxia, sometimes referred to as "leans" or "staggers" (Fregly, 1974; Fregly & Kennedy, 1965). Other signs (cf., Colehour & Graybiel, 1966; Mone, 1970; McClure & Fregly, 1972) include changes in cardiovascular, respiratory, gastrointestinal, biochemical and temperature regulation functions. Other symptoms include general discomfort, apathy, dejection, headache, stomach awareness, disorientation, lack of appetite, desire for fresh air, weakness, fatigue, confusion and, occasionally, incapacitation. The consequences for human performance and operational efficiency are decreased spontaneity, carelessness and incoordination, particularly in manual control. Motion sickness is theoretically preventable, but that is not always practical. Once symptoms become severe, treatment other than time may be impossible for subsidence.

# SECTION II

## STIMULUS CHARACTERISTICS

Many types of motion produce motion sickness. Among the most common places for sickness to occur are: 1) ships (Kennedy et al., 1968; Wiker, Pepper & McCauley, 1980) and small boats (Kennedy & Graybiel, 1962); 2) cars (Chinn, 1963), trains (Kaplan, 1964), and other motor conveyances; 3) gliders (Chinn, 1963), aerobatic (Kennedy & Graybiel, 1962) and zero gravity aircraft (Kellogg, Kennedy & Graybiel, 1965); 4) rotating rooms (Graybiel, Kennedy, Knoblock, Guedry, Mertz, McLeod, Colehour, Miller & Fregly, 1965) and chairs (Kennedy & Graybiel, 1965); 5) vertical oscillators (Alexander, Cotzin, Hill, Ricciuti & Wendt, 1945a, b, c, d; O'Hanlon & McCauley, 1974; Guignard & McCauley, 1982); 6) horizontal swings (Hemingway, 1942, 1946); 7) moving base (Kellogg, Castore & Coward, 1980; Crosby & Kennedy, 1982) and fixed base (McGuinness, Bouwman & Forbes, 1981) flight simulators. In addition, elephants and camels, but not horses (Money, 1970), tilted rooms (Witkin, 1949), buildings and chimneys (Irwin, 1977) have been implicated.

Linear oscillations vertically (i.e., parallel to the long axis of the body) are generally considered to be the most debilitating, but horizontal motions (generated by two- and four-pole parallel swings) were the methods of choice in studying air sickness in World War II by the USAF and the RAF (Hemingway, 1942, 1946; Joekes, 1942). Rotary motions produced by carnival devices and centrifuges also are very effective in producing motion sickness (Kennedy & Graybiel, 1965). Complications of these stimuli tend to produce motion sickness more effectively (Graybiel & Miller, 1970; Guignard & McCauley, 1982) and there is a suggestion that with the head free to move there is more sickness than with the head fixed (Johnson, 1952; Johnson & Mayne, 1953).

Humans appear to be most susceptible to motion sickness when exposed to very low frequency vibrations in the range of .12 - .25 Hz (McCauley & Kennedy, 1976), although these data are limited largely to those from swings and vertical oscillators. There does not appear to be this frequency specific relationship when cross-coupled angular accelerations are employed (Kennedy & Graybiel, 1965). The normal locomotor behavior of man has a mean frequency of about 1.7 Hz (Ashley, 1970; Rao & Jones, 1975) and, as mentioned, does not induce sickness. Most of the power in spectral density analyses of body sway is below .40 Hz (Bensel & Dzendolet, 1968) and perhaps platform stimuli in this range could be amplified at the head.

Although acceleration of the environment is generally required, visual motion alone is sufficient to produce sickness (Crampton & Young, 1953; Dichgans & Brandt, 1973). The effects are usually limited to the period of exposure, but postadaptation " effects are known to occur (Fregly & Kennedy, 1965; Witkin, 1949; Reason & Brand, 1975) and in some cases (Lackner, 1984, personal communication) using optokinetic stimulation with a stationary observer, the symptoms begin to develop some time after the subject has left the test situation. This has also occurred when persons have reported complete incapacitation for up to 24 hours following an hour or two hiatus after Slow Rotation Room exposures (Kennedy, 1961, unpublished observations). Although not experimentally verified, it is rumored that labyrinthine defective (LD) individuals, while stationary, do not experience discomfort when exposed to moving visual fields which induce symptoms in normal vestibular functioning people.

# SECTION III

# RESPONSE CHARACTERISTICS

## BEHAVIORAL

Along with the signs and symptoms of motion sickness listed in the definition section of this paper, there are several noteworthy responses which are likely to have an influence on performance.

ATAXIA. Motion-induced vestibular ataxia is not widely known, but has been reported following protracted exposures to a centrifuge and to ships at sea (cf., Fregly, 1974, pp. 338-346). Data are available whereby ataxia performances due to elevated blood alcohol levels were used as a basis for comparison to posteffects in a U.S. Navy ground-based flight simulator (Crosby & Kennedy, 1982). Under ordinary circumstances, spectral density recordings of body sway (Bensel & Dzendolet, 1968) show the greatest power at around .20 Hz. Because there are considerable individual differences in power spectral density functions (and they appear to be reliable signatures of persons) it is possible that some aspect of an individual's frequency response may relate to his ability to acquire adaptation or "sea legs." Both postural equilibrium and steering or tracking are closed-loop psychomotor control systems under voluntary guidance by the cerebral cortex and under automatic (i.e., motor) control in the cerebellum (Hill, 1971; Stockwell & Koozekanani, 1981). Thus, it is not unreasonable to expect that if posture is disrupted (in the form either of a bias or increased variance) by exposure to motion, human manual control may be similarly affected (Adams, 1977; Angel, 1976; Cohen, 1970b). The motion-induced ataxia may be analogous to eye-hand coordination changes following rearranged visual feedback which occurs when wearing reversing or displacing prisms (Cohen, 1970a, b; Held, 1965; Jones, Davies & Gonshor, 1977; Jones, 1983; Stratton, 1896, 1897a, b, c).

DROWSINESS/SLEEP LOSS/SOPORIFIC EFFECTS. It is well known that a cardinal symptom of motion sickness is drowsiness (Graybiel & Knepton, 1976). Related to this are the findings that the mesencephalic reticular formation and vestibular nuclei mutually modulate each other, probably through the dense fiber pathways of the cerebellum (Yules, Krebs & Gault, 1966). It might be inferred that the drowsiness, which anecdotally is known to follow train trips, bus trips, and aircraft flights, also is an aftereffect of motion and produces fatigue.

The soporific effects of motion are known to all who have observed the calming effects to infants, pets, and others when

embarked on long car trips or train rides. Less well known is the operational or industrial consequence of this same drowsiness induced by motion (Graybiel & Knepton, 1976). Although their work schedules are demanding, greater than average sleepiness definitely occurs at sea, as any career seaman will attest. Woodward and Nelson (1976) have described the types of performance impairment most likely from general sleep loss, including slower reaction time, short-term memory decrement, impairment in reasoning and complex decision making, error of omission, and lapses of attention. It is possible that the drowsiness that often accompanies vestibular upset may have similar effects on human performance. Lackner (1984, personal communication) is of the opinion that transient personality changes may be associated with the sopite syndrome.

Sleep loss has also been shown to have a deleterious effect on vestibular processes. Dowd (1974) reported increased vestibular sensitivity, decreased recovery rate, and abnormal vestibular habituation to be associated with sleep deprivation. He warned of the implications of sleep loss for increasing the hazards of flying due to degraded vestibular function. The vestibular system is considered to be under control of the pontine reticular formation (Yules, 1967; Yules et al., 1966). The connection of the vestibular system with sleep and electroencephalograms, while not widely known, is also not a new concept (cf., Lindsley & Wendt, 1944; Collins, Crampton & Posner, 1961; Collins & Posner, 1963).

AFTEREFFECTS. The incidence and magnitude of motion aftereffects in Naval personnel after disembarking is not known. However, it is known that accidents involving personal vehicles during off-duty hours are the greatest cause of injury and death to Naval personnel during peace time. It is possible that motion aftereffects contribute to these and other accidents.

PERFORMANCE. Little data exist on the effects of motion sickness on human performance, per se. This lack is due, in part, to the difficulty in producing the appropriate bandwidth of very low frequency vibration (VLFV) in the laboratory to induce sickness along with the problem of collecting data in operational settings. For example, a sinusoidal motion at .10 Hz and .10 peak g requires a full-wave vertical displacement of over 16 feet. McCauley and Kennedy (1976) have reviewed these problems elsewhere; the following is extracted from that report (pp. 4-5).

In a comprehensive review of low-frequency motion and human performance, Baker (1966, p. 2) commented "...there is virtually no pertinent, documented information regarding the effects of either motion sickness or of motion upon human performance." The common finding is that task performance

simply ceases in conjunction with the cardinal sign of motion sickness (i.e., vomiting). More subtle evidence of performance decrement prior to sickness has not been consistently found. An exception is the increased tracking error obtained when the low-frequency motion exerts a direct biodynamic interference with the task. These effects represent complex interaction among the motion, the control dynamics, the displays, and the man (Hornick et al., 1972; Jex & Allen, 1974), and are not amenable to one set of exposure limits.

In a series of studies on VLFV conducted by Professor G. R. Wendt and his associates during the 1940s (Alexander, Cotzin, Hill, Ricciuti & Wendt, 1945a, b, c, d; Alexander, Cotzin, Klee & Wendt, 1947), pychomotor performance tests were investigated, including an obstacle course, a 60-yard dash, dart-throwing, and the Mashburn Complex Coordinator (a tracking device used in pilot selection). These subjects, mostly U.S. Navy Cadets, performed the psychomotor tests before and after a 20-minute exposure to a motion of a vertical accelerator similar to an elevator. Pretest and posttest performance scores of subjects who became motion sick were compared with scores of subjects who did not. Results showed virtually no effects of motion sickness on the performance tests (Alexander et al., 1945d; Baker, 1966).

A direct and comprehensive investigation of the effects of low-frequency motion on performance was done by Abrams, Earl, Baker and Buckner (1971). Naval personnel performed a variety of perceptual, psychomotor, cognitive, and simulated operational tasks during 64-hour missions in a three-degree-of-freedom (heave, pitch, and roll) sea motion simulator. The dynamic environment simulated the motion of a hypothetical air/seacraft, dead in the water, in sea states 3 through 5. Data for a control condition were collected simultaneously from subjects in an identical, though stationary, cabin. Subjects in the dynamic condition showed no systematic performance decrements on any of the tasks, except when they became physically incapacitated due to vomiting. A field study of motion sickness and performance was reported by Kennedy, Moroney, Bale, Gregoire and Smith (1972). They measured performance on a counting/short-term memory task in three types of large aircraft undergoing severe turbulence during hurricane penetrations. The main finding was that performance decrements were directly related to amount of turbulence experienced; whereas the incidence of motion sickness appeared to be only partly correlated with turbulence and partly with the periodic frequency of the motion. Because motion sickness symptomatology did not correlate well with performance degradation, it is probably safe to infer that these two behavioral problems derive from different mechanisms. In a laboratory study with the same task, no performance decrement was found when subjects were subjected to angular

oscillation about the Z-axis (Kennedy, 1972). In other studies of performance in rotary environments, similar results have been found, namely no systematic performance decrements (Guedry, Kennedy, Harris & Graybiel, 1964). Notable exceptions are the loss in visual acuity and tracking problems associated with vestibular nystagmus when the visual target is fixed with reference to the subject (Guedry, 1968; Gilson, Guedry & Benson, 1970), and increased error in time estimation during a rotation at 10 rpm (Graybiel, Kennedy, Knoblock, Guedry, Mertz, McLeod, Colehour, Miller & Fregly, 1965).

Money (1970) lists other effects of motion sickness on performance not mentioned above, namely: 1) decreased spontaneity, inactivity, or being quiet or subdued; 2) carelessness in the performance of duty; 3) decreased muscular coordination; 4) decreased tracking performance; 5) decreased squeezing ability; 6) decreased time estimation; 7) decreased arithmetic computational ability, and no effect on arithmetic computational ability. There have also been studies which did not obtain degradation on: 1) arithmetic, 2) vision tasks, 3) dart throwing, 4) mirror tracing, and 5) rifle shooting.

PHYSIOLOGICAL

BIOCHEMICAL. Table 1 (adapted from Nicogossian & Parker, 1982) summarizes the physiological responses that occur as a function of motion sickness.

ADAPTATION/HABITUATION. Humans are adaptable. The effects of almost any environmental stressor on performance and physiology will change over time (duration of exposure). The nature of the change is usually that the observed effect diminishes with time. These generalizations obviously have limits, since extremely intense stressors can cause injury or death (precluding adaptation). Predictions of performance decrements, ataxia, or other potential effects due to motion are difficult to make because the extent and time-course of adaptation are not known, and may only be inferred from the visual distortion literature (Kennedy, 1970).

There are both large individual differences in adaptation and large time-course variances within an individual's adaptive process to differing motion environments. Recently, however, Graybiel and Lackner (1983) have shown that individuals appeared to adapt in a similar manner to three different provocative motion environments.

Moreover, adaptation is a double-edged sword. It implies a modification of sensory processes to enable the individual to function more successfully in the presence of an altered environment. When the individual returns to his "normal" environment, however, the modified sensory processes most

TABLE 1. PHYSIOLOGICAL MOTION SICKNESS MANIFESTATIONS

| Physiological System | Manifestations |
|---|---|
| Cardiovascular | Changes in pulse rate and/or blood pressure.<br>↑tone of arterial portion of capillaries in the fingernail bed.<br>↓diameter of retinal vessels.<br>↓peripheral circulation, especially in the skin of the head.<br>↑muscle blood flow. |
| Respiratory | Alterations in respiration rate.<br>Sighing or yawning.<br>Air swallowing. |
| Gastrointestinal | Inhibition of gastric intestinal tone and secretions.<br>Salivation.<br>Gas or belching.<br>Epigastric discomfort or awareness.<br>Sudden relief from symptoms after vomiting. |
| Body Fluids, Blood | Changes in Lactic Dehyorogen ASE concentrations.<br>↑hemoglobin concentration.<br>↑pH and ↓$paCO_2$ levels in arterial blood, presumably from hyperventilation<br>↓concentration of eosinophils.<br>↑17-hydroxycorticosteroids.<br>↑plasma proteins.<br>↑ADH.<br>↓Glucose utilization. |
| Urine | ↑17-hydroxycorticosteroids.<br>↑catecholamines. |
| Temperature | ↓body temperature.<br>Coldness of extremites |
| Visual System | Ocular imbalance.<br>Dilated pupils during emesis.<br>Small pupils.<br>Nystagmus. |

Adapted from Nicogossian & Parker, 1982.

probably will not be optimal. The adaptation must occur in the opposite direction (readaptation) for the individual to function optimally in his normal environment. This type of adaptation/readaptation process has been well documented in the research literature under a variety of environmental influences such as optical distortion (Baily, 1972; Bossom, 1974; Burnham, 1968; Day & Wade, 1966; Denton, 1977; Ebenholtz, 1969; Gibson, 1937, Held, 1965; Kennedy, 1970; Lackner, 1973; Over, 1970),weightlessness (Kellogg, Kennedy & Graybiel, 1965; Lackner & Graybiel, 1980), rotation (Graybiel et al., 1960; Guedry et al., 1964; Guedry, 1965; Graybiel, Clark & Zarriello, 1960), and rectilinear motion (Denton, 1977).

It is very likely that adaptation, in the form of less symptomatology during repeated simulator exposure, will occur to the perceptual rearrangement found in flight simulators (whether visual or inertial). HOWEVER, TO RELY ON THEIR REDUCTION OR ELIMINATION THROUGH ADAPTATION MISSES THE POINT OF THE REQUIREMENT FOR MINIMUM HUMAN FACTORS ENGINEERING DESIGN CRITERIA, AND MAY ALSO IMPACT ON SAFETY OF SUBSEQUENT FLYING AND OTHER ACTIVITIES. The very adaptation that reduces the effects during exposure to the simulated environment may cause problems when the person returns to the normal environment. Furthermore, these effects may interact in peculiar ways, should the individual be transported in a conveyance, either under his own control (e.g., a car), or not.

# SECTION IV

## ANATOMICAL STRUCTURES RELATED TO MOTION SICKNESS

This section builds upon Money and Wood's (1968) excellent review of the neural mechanisms underlying the symptomatology of motion sickness.

### VESTIBULAR APPARATUS

The variety of the physiological responses presented in Table 1 suggests that motion sickness symptoms are not the discharge from a single source and point out the complexity of the malady. It is, therefore, difficult and may be impossible to identify specific anatomical structures and relationships that will always be implicated in motion sickness. Fortunately, however, one finding in the motion sickness literature remains constant: only individuals with a normally functioning vestibular system can become motion sick. This has been implied since James (1882), but was provocatively brought home in a series of studies with labyrinthine defective subjects, directed by Graybiel and his associates, and culminated with exposures to storm conditions in the North Atlantic, in a round-bottomed tug lacking stabilization gear (Kennedy et al., 1968).

The vestibular apparatus (or labyrinth) can be divided into two functional components: 1) the semicircular canals, the receptors for angular acceleration, and 2) the otoliths (housed in the utricle and saccule), the receptors for linear accelerations. Whether it is the canals, the otoliths, or both, that are the requisite structure(s) for motion sickness is unclear (Money & Cheung, 1983). This is because it is difficult to perform experiments that unequivocally isolate the structure of interest.

Money and Friedberg (1964) have shown that the plugging of the semicircular canals (rendering them inoperable, but leaving the otoliths functional) of dogs was as effective in eliminating sickness as bilateral labyrinthectomy. However, it has been suggested that this stimulus was a two-pole swing. In such a device, the major stimulus is to the semicircular canals while the resultant linear vector remains aligned with the z-axis. Therefore, a canal-otolith conflict results which may be lost when the canals are plugged. Benson (1974) and others have shown that the canals may also respond to changing linear acceleration, thereby confounding the data ascribed to demonstrate otolith involvement in motion sickness. Money's (1970, p. 14) assessment of a decade ago is still valid today, "In summary, it appears that the vestibular apparatus is indispensable for the occurrence of motion sickness and that

under certain circumstances the semicircular canals are indispensable; the otolith organs may also be indispensable."

VISUAL APPARATUS

As mentioned earlier, visual motion alone is sufficient to induce motion sickness (e.g., Crampton & Young, 1953; Dichgans & Brandt, 1973). Several fixed-base aircraft simulators are known to induce motion sickness symptoms, although emesis is seldom reported (Frank, Kennedy, McCauley & Kellogg, 1983), except in one case which occurred at the Naval Training Equipment Center's Visual Technology Research Simulator. On this occasion, the pilot vomited in the simulator but he reported that he had also just swallowed his tobacco juice. It is tempting to speculate whether the swallowed tobacco juice was of a sufficient emetic dosage to have resulted in sickness all by itself. Alternatively (following the poison theory discussed later), perhaps a certain amount of juice can be added to a certain amount of simulator conditions to produce vomiting. If verified, it would suggest that hangover, flu, and other ailments could also summate with simulator aspects to contribute to the incidence of simulator as well as other forms of motion sickness.

In general, it appears that a large field of view or a large field of movement is required to induce sickness. However, a systematic exploration of motion sickness symptomatology as a function of field of view has not been performed.

Casali and Wierwille (1980) have pointed out another potentially salient factor. In a study of sickness in driving simulators, it was found that symptomatology was greater when an enclosure (simulating a cab) was positioned around the simulator. The present authors have noted that in flight simulators, when structural features of the room housing the simulator are visible to the person in the simulator, or when artifacts (like lens imperfections or scratches) are present, then symptoms of discomfort are lessened. It is our guess that appurtenances interact, although perhaps not in conscious ways, so that the kinematics are somehow less compelling.

Exactly how the visual and vestibular systems interact is not known. The perceptual conflict theory of motion sickness (Reason, 1970, 1978), as we shall see later, would predict motion sickness when the visual and vestibular systems are in conflict. The simplicity of this explanation is intriguing but, as will be seen, it may also be an oversimplification.

VISCERA

Because the viscera move, some have thought such movement may cause the nausea and vomiting associated with motion

sickness (Irwin, 1881). Money and Wood (1968) have shown that denervation in the viscera of dogs did not markedly reduce susceptibility. Moreover, sympathectomy, vagotomy, or both, had no effect on susceptibility.

PROPRIOCEPTION AFFERENTS

The nonvisual proprioceptive afferents may play a role in motion sickness, but its effects are unknown (Money & Wood, 1968).

AFFERENTS FROM THE EYES

Vision has a role, not only in potentiating the effects of motion sickness (e.g., when visual-vestibular conflict is present), but also in reducing sickness (e.g., a fixed external reference can reduce the incidence of sickness produced by some motions). However, blindfolded (Kennedy, Tolhurst & Graybiel, 1965) and blind (Graybiel, 1970) subjects can be made sick.

PERIPHERAL EFFERENTS

Money and Wood (1968) feel that the gamma efferents may somehow sensitize the vestibular inputs. They point out that the important nerves for vomiting are the phrenic nerve of the diaphragm and the spinal nerves to the intercostal and abdominal muscles. Nausea, which can be experienced after total gastrectomy, is probably the conscious awareness of unusual activity in vomiting centers, rather than autonomic activity. Pallor and cold sweating, however, are very likely autonomic phenomena, although they could also be due to a circulating chemical (Crampton & Daunton, 1983).

VESTIBULAR NUCLEI

According to Money and Wood (1968), it is probably necessary that these neural complexes be intact. However, the four main constellations of vestibular nuclei are intermingled along with other cells and tracts. In this region, there are multiple-crossed and uncrossed connections, and the area postrema (i.e., chemoreceptor trigger zone) is proximate to these nuclei and the IVth ventricle. Because this area of the brain stem contains structures with complex and different functions, and because there are feedback loops within feedback loops, it is very difficult to perform the necessary experiments to establish the requirement for their presence. Much of the work in this area continues to be largely neuroanatomical rather than physiological. Exceptions seem to be the recent work of Crampton and Daunton (1983a, b).

VESTIBULAR PARTS OF THE CEREBELLUM

The uvula and nodulus are necessary; the folium and tuber vermis are probably important (Wolfe, 1966), particularly for

habituation, and perhaps for all other classes of response modification (i.e., adaptation).

## CHEMORECEPTOR TRIGGER ZONE

The chemoreceptor trigger zone lies in the area postrema of the fourth ventricle. This mechanism is definitely necessary for the vomiting reflex to occur to motion (Wang & Chinn, 1954).

## CEREBRUM

The cerebrum does not appear to play a vital role, although it may be the source of motivation loss, discomfort, lassitude, and other problems. Decorticate man has been reported to become sick.

## LIMBIC SYSTEM

Kohl (1983) believes that the neural mismatch center lies in the limbic system. His article offers much speculation, mostly in the form of allusions to what goes on neurobiochemically, but we believe the evidence is circumstantial. It is well known that stimulation of the vestibular system results in innervation of the vagus nerve with the consequences that multiple autonomic nervous system structures will also be affected, including the limbic system. Others have suggested that the coordinating center is in the cerebellum (Kennedy, 1970; Snider, 1958), and there is some circumstantial evidence to support that view. More research is needed in this area.

# SECTION V

# OTHER FACTORS

## INDIVIDUAL DIFFERENCES

People differ in everything, and dramatic dispersions are evident in susceptibility to motion effects. The only people who are immune are persons with defective organs of equilibrium (labyrinthine defectives), and who are generally deaf due to damage or disease to the VIIIth cranial nerve (Kellogg et al., 1965; Kennedy et al., 1968). It is probably a good assumption that people are normally distributed in terms of their susceptibility to motion effects. Data from studies of motion sickness suggest the assumption of a normal distribution of susceptibility in college-age males (McCauley et al., 1976).

Individual differences in adaptability also exist (Barrett & Thornton, 1968; Gibson, 1937; Reason & Brand, 1975; Witkin, 1949). Money (1970) has reported that most of the population will adapt to motion sickness through repeated exposures to a moderate motion environment. Conversely, some persons do not adapt. Such people clearly would self-select out of work in a unsuitable motion environment. However, the role of adaptation is not clear. In one laboratory study, McCauley et al. (1976), set out to study habituation to distributed exposures and did not find sufficient evidence to support such a notion, with a small sample exposed to linear oscillations.

## SEX

Women are more susceptible to motion sickness than men; precisely why is unknown. It has been postulated that perhaps hormonal influences are at play, since women are most susceptible during their menstrual cycle (Schwab, 1954). Also noteworthy is that women exhibit larger fields of view than men, from the standpoint of functional peripheral fields (Burg, 1968, cited in Leibowitz, 1973). As previously noted, simulator sickness appears more prevalent in simulators with wide fields of view (Frank et al., 1983).

## AGE

The distribution for susceptibility to motion sickness as a function of age is negatively skewed. Susceptibility is highest for those individuals between roughly two years of age and puberty. There is a rapidly decreasing susceptibility between puberty and about 21 years, with susceptibility decreasing gradually thereafter, and almost disappearing after age 50 (Benson, 1978).

## FIELD DEPENDENCE/INDEPENDENCE

Perceptual style appears to be a factor related to motion sickness. Field-dependent individuals are influenced more than field-independent people by the context in which a perceptual judgement has to be made (e.g.,embedded figures; rod & frame). In studies of simulator sickness it has been found that field-independent people were more susceptible to simulator sickness than field dependent (Barrett & Thornton, 1968; Barrett, Thornton & Cabe, 1970; Testa, 1969).

Barrett and Thornton (1968) attempted to explain the relationship they obtained between the differing perceptual styles and sickness through the perceptual conflict (mismatch) theory of motion sickness. According to Barrett and Thornton, field-independent people are more sensitive to body cues. Hence, when they are placed in a fixed-base simulator that has conflicting visual information (i.e., movement) sickness arises. Field-dependent individuals on the other hand, may be more resistant to the conflict.

Earlier, it was noted that women are more susceptible to motion sickness then men. Interestingly, women are also more field dependent than men (Witkin, Lewis, Hertzman, Machover, Meissner & Wapner, 1954). Ebenholtz (personal communication, 1984) has shown that the size of the rods and frames can influence whether an individual obtains scores indicating he is field dependent or field independent. It is obvious that the relationship between field dependence/independence and motion sickness is not simple.

## HEAD MOVEMENTS

It has been shown that head movements increase motion sickness susceptibility in gliders, slow rotation rooms, and swings (Johnson, 1952; Johnson & Mayne, 1953;). Similarily, it has been reported that increased head movements during space flight are correlated with space motion sickness (Matsnev, Yakoleva, Tarasov, Alekseev, Kornilova, Mateev & Gorgiladze, 1983; Graybiel, 1979). Likewise, they have been implicated as elements which may contribute to simulator sickness (Sinacori, 1969).

It is also known that the supine position greatly reduces the incidence of motion sickness for swing-type movements (e.g., ships, trains). It may be that the restricted head movement incurred when lying down, or the reorientation of the labyrinth relative to the direction of motion, is germane--and possibly both are.

## PREDICTABILITY

The ability to predict a person's susceptibility to motion sickness, from one motion environment or set of stimulus

conditions to another, is poor (Reschke, Homick, Ryan & Moseley, (1984). We believe it is because the reliability of the criterion for assessment of motion sickness susceptibity may be low. Wendt (1968) noted that an individual history of vomiting accounted from 4 to 35% of the variance of motion sickness. Hence, the lability of the motion sickness control system (perhaps analogous to the blood pressure control system) may be large relative to the between-subject differences. Thus, it may be difficult to predict susceptibility, except by averaging over several exposures--an obvious experimental design problem for the space program.

Indeed, this factor is considered to be a chief limitation to the field of study into motion sickness. Firstly, it is unpleasant to be made sick even once, and it is arguable whether in a second measure one may avoid carryover effects from the first, a usual requirement for repeated measures. statistical methodologies. Nonetheless, it is essential to obtain replicated measures of susceptibility in order to retain sufficient power to undertake small sample studies. It is obviously unethical to employ more subjects than are necessary for studies where discomfort is induced, and so the whole question of experimental design is a difficult one to handle satisfactorily. It is our view that a new look needs to be taken for purposes of identifying the approach to be employed in prediction.

# SECTION VI

# THEORIES OF MOTION SICKNESS

## OVERSTIMULATION THEORY

Probably the oldest theory of motion sickness (McNally & Stuart, 1942) is the overstimulation theory. This theory simply states that motion sickness is caused by intense stimulation of the vestibular system. Thus, the overstimulation theory is specific to the vestibular system and predicts that as stimulation increases, the likelihood or severity of sickness increases. The genesis of the overstimulation theory may go back to the nineteenth century, when Irwin (1881), a ship's physician, noticed that deaf persons were immune to seasickness. He concluded that since congenital damage to hearing could be accompanied by partial destruction of the auditory nerve subserving semicircular canal functioning, perhaps the angular accelerations at sea occasioned an irritation in normal persons' semicircular canal systems, and that this was the cause of motion sickness.

There is considerable evidence that overstimulation does not satisfactorily account for all incidences of motion sickness. As has been mentioned, vision alone is sufficient to induce sickness as demonstrated in the case of some fixed-base simulators (e.g., Frank et al., 1983). Motions that are difficult to view as overstimulating, such as slow rotation rooms and ship movement, can induce severe sickness. Moreover, many intense motions, such as aerobatic flight and mechanical bucking broncos, do not necessarily cause sickness. It is true, though, that the incidence of motion sickness generally increases with stimulus energy (e.g., acceleration and changes in acceleration), but the chief exception to this is the seeming frequency specific relationship, as with .20 Hz in linear oscillation.

It appears then that the overstimulation theory may only be partly correct as an explanatory principle for motion sickness.

## FLUID SHIFT THEORY

The idea that fluid shifts within the body may contribute to motion sickness is new (Steele, 1968) and old (Wollaston, 1811). The latter claimed that motion sickness was caused by the sloshing of the blood such that there was alternate engorgement and anemia of the brain.

At first glance, the literature on fluid shift theory appears to divide itself into contradictory camps. The first stating that motion sickness is caused by the inadequacy of

cerebral circulation; the second, that it is caused by an overabundance of cerebral circulation. These diametrically opposed views may be resolvable, because the "inadequacy camp" is addressing motion sickness on earth, whereas the "abundance group" is addressing motion sickness in space flight.

INADEQUACY OF CEREBRAL CIRCULATION. According to Steele (1968, p. 93), "...the most severe motion sickness symptoms seem to be caused by a decrease in circulating blood volume." Steele points out that there exists a high correlation between susceptibility to motion sickness and the liability of cerebral circulation on a horizontal swing (Van Egmond, Groen & deWit, 1954).

Johnson and Hsuen (1970) measured brain-blood flow changes in dogs. Their procedure consisted of implanting thermistors in the thalamus and using a two-pole swing as the provocative stimulus. Increases in thalamic temperature were interpreted as decreased blood flow to the brain. Their data showed that initially thalamic (brain) temperature fell as the swing was set in motion and continued to gradually decrease (increasing cerebral flow) until immediately prior to the act of vomiting, at which time the temperature rose.

Steele (1968) also believes that the reason that .20 Hz is the optimal frequency for inducing motion sickness is that it is "...approximately the natural slosh frequency of the blood" (p. 92). Alternatively it may be that there are "natural frequencies" around .20 Hz for parts of the vestibular system (McCauley, et al., 1976; Robinson, 1968).

Perhaps the major problem with the inadequacy of cerebral circulation is that blood flow changes may be a result of motion sickness and not an antecedent condition. Decreased cerebral blood flow could very easily be a response initiated by the chemoreceptor trigger zone or be a result of channeling blood to the viscera to assist in the job of emptying the stomach contents.

In brief, there is a paucity of data to support the inadequacy of cerebral blood flow fluid shift theory of motion sickness.

OVERABUNDANCE OF CEREBRAL CIRCULATION. By far, the majority of research and interests in fluid shift theory has derived from its possible explanation for space motion sickness. During space flight there is a cephalic shift of 1.5 to 2.0 liters from the lower extremities (Nicogossian & Parker, 1982). Calf girth correspondingly decreases about 30 percent. Mean resting heart rate and systolic blood pressure tend to increase, while diastolic pressure decreases.

According to space fluid shift theory, the rostral shift in body fluid alters cranial pressure and vestibular response. For example, altered fluid pressure in the labryinth could result in a change in gain and phase shift (Wolfe, Engelken & Olson, 1981, cited in Parker, Tjernstrom, Ivarssen, Gulledge & Poston, 1983). Graybiel and Lackner (1977, 1979) have examined the evidence for this theory on earth by the use of head-down tilt to induce fluid shift. Their work has shown that fluid shift towards the head has either no effect on susceptibility or a small decrease in susceptibility as the magnitude of the shift increases (Lackner & Graybiel, 1983).

In our judgment, fluid shift theory appears to be a weak theory, not only for simulator sickness, but also for earthly and space sickness, although fluid shifts could perform some modulating influence on vestibular threshold functions.

## FEAR/ANXIETY THEORY

Does anxiety or fear increase a person's susceptibility to motion sickness? According to Benson (1978, p. 486), "...a definite correlation between susceptibility and psychometric measures of anxiety or neuroticism has not been established." It is not known for certain whether this is due to a true lack of relationship, or perhaps to the lack of reliability in measures of anxiety (Kennedy, 1972), as well as to the already mentioned lack of reliability in measures of the motion sickness criterion. In any case, clear-cut evidence for the notion is hard to find, in spite of the fact that such a relationship is intuitive, neo-Freudian and neo-Pavlovian! Although there are no hard data to support the supposition that anxiety is a provocative stimulus for motion sickness, psychiatric data are suggestive. Reinhardt (1968), in his work as a military psychiatric flight surgeon, strongly believed that anxiety was a primary factor responsible for the air sickness seen in initial flight training. Anecdotal data suggest that anxiety does increase susceptibility. For example, one of the authors is convinced that anxiety was responsible for the two occasions he experienced nausea as an aircrewman in a tactical jet. (The other author is just as certain that his sickness at sea, but never in aircraft or other moving vehicles, is not related to anxiety.) The role anxiety plays in motion sickness is nebulous, but efforts to examine its relationship to motion sickness should continue.

## BALANCE OF AUTONOMIC ACTIVITY POSTULATE

Waxing and waning of symptoms suggests competing processes (Wood, 1970). The symptoms of motion sickness resemble what one might associate with increased cholinergic activity (Koelle, 1965) and decreased adrenergic, but the relationships are not clear-cut (Tang, 1970). While the drugs which are effective in

motion sickness are chiefly those which stimulate the sympathetic nervous system, or those which shut down the parasympathetic nervous system, the several exceptions (Tang, 1970) imply that this postulate should be considered as part of a larger theory.

## TOXIC REACTION THEORY

Treisman (1977) addressed the evolutionary significance of the emetic response to motion sickness. What, Treisman asked, is the adaptive function of vomiting during motion sickness, and how does such a response contribute towards the survival of the species? His answer was that the only adaptive significance vomiting could have is the explusion of ingested toxins from the body. Hence, when the body vomits in response to motion sickness, it is interpreting the stimulus as if it were a poison. Wiker (1981) has also made this point.

Normally, the sensory systems of the body complement each other. The eyes and the vestibular system are in harmony. When a toxin is ingested, it reacts on the inner ear causing the vestibular signal to come in conflict with vision and other senses. This conflict signals the body that it has ingested a poison and emesis occurs.

Treisman eloquently "...points to the differences found in motion sickness susceptibility associated with age and sex and attributes such differences to food gathering activity. Infants, who do not search for food and rely upon breast milk, are not generally susceptible to motion sickness. Adolescents, who are not likely to be skilled in food selection, or aging adults with failing near vision, are generally more susceptible to motion sickness than young adults. Women, who breast feed and who traditionally have been charged with the selection and gathering of food, appear more susceptible than males." (Wiker, 1981, p. 17.)

In order to test Treisman's theory, Money and Cheung (1983) performed bilateral labyrinthectomies on dogs after establishing vomiting thresholds to four poisons. Following labyrinthectomy, it was found that for some poisons the time to vomit increased or that no vomiting occurred, although for some animals and some poisons vomiting continued to occur. This implies that the vestibular system is implicated in the poison pathway. Alternative interpretations are that central nervous system integration of canal/otolith/visual and proprioceptive inputs to the vestibular nuclei and vestibular cerebellum are influenced by toxins.

## PERCEPTUAL CONFLICT THEORY

Perceptual conflict theory is known by several names: mismatch, neural mismatch, cue conflict, incongruity and

sensory rearrangement being the most common. The present authors believe that perceptual conflict is the most descriptive term and, consequently, recommend its use here.

In brief, the perceptual conflict theory posits a referencing function in which motion information, signaled by the eyes, vestibular apparatus or proprioception, may be in conflict with these inputs' expected values, based on a neural store (which reflects past experience) or with how the system circuitry is wired (i.e., naturally endowed).* Kennedy (1970) suggested, as have others (Held, 1965; Reason, 1970), that perceptual conflict theory is based on a lack of correlation between appearance and reality. Under ordinary circumstances, there is a correspondence between what is sensed and the physical representation of the stimulus. The sensory systems report reality, and after periods of time, create a neural store of expectations. The expectations are also referenced to the sensory channel which delivered them, being stronger for more experiences and also in those ranges where the channel is most sensitive. The purpose of information processing and perception functions is to predict reality in order that one may interact with it, spatially and temporally. We believe that central nervous system integration could be represented by a linear model (Cohen, 1968).

For our version of the conflict theory, the following working principles are meant to hold, with respect to conflict, in the following examples: 1) acquired percepts achieve their strength (or salience) through feedback repetition, and the usual generalities from the literature of learning and retention apply; 2) endowed percepts achieve their strength according to their sensitivity to detect physical energy, and salience is proportional to what the literature reports as minimal and difference thresholds for the stimuli involved; 3) based on our evaluation of 1 and 2 above, we suggest that ordinarily visual information is likely to be more salient than information delivered over other pathways; 4) greater conflict occurs when the percepts which are not in accord are more rather than less salient; 5) the less agreement between the two inputs, the greater the conflict; 6) salience can be similarly quantified, whether derived from endowed or acquired percepts; 7) conflict may occur between disparate spatial arrangements of stimuli, temporal arrangements of stimuli, or both; 8) conflict between sensory information pathways most commonly occurs spatially, but can occur temporally when time delays occur within a pathway; 9) conflicts can occur between temporal and spatial inputs.

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*Although not popular these days, it is still possible for one to adopt either an extreme "nature" or "nurture" theory of perception. What follows implies a combination theory, but the relationships which are discussed ought to work in either case, regardless of which emphasis is taken.

The following principles are meant to hold, with respect to adaptation, in the examples described below: 1) correlations occur between and within sensory pathways, and may be between spatial or temporal information, or both; 2) adaptation occurs more quickly when correlations are high (even though negative); 3) adaptation occurs more slowly when the salient pathways are incorrectly interpreting reality and the less salient are "right"; 4) adaptation is more rapid when there is fed back correlated information; 5) adaptation does not occur when correlations are zero or continually changing.

The three elements which appear in the above principles are salience, conflict and adaptation. We believe that conflict may be likened to predictive validity. We believe that salience may be likened to reliability. Adaptation is a time dependent (repeated measure) construct. In general, the greater the conflict and the less the adaptation, the greater the sickness, particularly among salient stimuli. The magnitude of the symptoms can change (adaptation) either because conflict or salience change. All three of these interact.

## SECTION VII

## EXPOSITION

Ordinarily, the various sensory inputs are in accord. I see, hear, and feel the relationships of my movement through space. However, occasionally this is not so. For example, when I can not see outside the cabin of a moving ship, my visual cues to motion are absent, while my vestibular and proprioceptive cues transmit combinations of motion information. If I were to be making predictions of reality from my inputs, I would discover that correlations between visual information and vestibular or proprioceptive were not perfect. If I continued to "trust" my visual input, possibly because of that pathway's long history of accurate portrayals of reality, I would incorrectly interpret physical reality. This, therefore, constitutes the "conflict." The strength of the conflict in this case is considered to be stronger than when proprioceptive or vestibular information is decorrelated because the visual inputs have more salience due to sensitivity and past history. Another way of quantifying salience is to suggest that high test/retest reliability occurs when measures are repeated and this same information is correlated with other sensory systems' samples of the same data obtained at the same time (i.e., high alternate form reliability). It follows that stimuli within certain bandwidths of frequency (or within certain ranges of acceleration) result in reliabilities which provide an index of the sensitivity of that sensory channel. If this were true, then disruptions or conflicts which occurred between sensory channels which were very sensitive in the ranges that were being stimulated would result in proportionally more conflict than when the two stimuli are outside the range of maximal sensitivity for one or the other pathway.

In correlation terminology, correlation coefficients can range from minus 1.00 through zero to plus 1.00. We propose to push the correlation model in an effort to derive a method for quantifying the magnitude of the conflict. For example, more sickness is expected to occur as a function of the conflict. Conflict is defined as a correlation which is less than 1.00. The magnitude of the conflict is proportional to the departure of the correlation from a perfect value. However, when conflicts occur, it is possible for the correlations to be slightly less than 1.00; or markedly less than 1.00; or even -1.00. In the latter case the conflict would be stronger, at least numerically, but it is also likely that continued stimuluation would result in improved adaptation because the conflict is so predictable. On the other hand, conflicts which are due to correlations around zero might never result in adaptation. When waveforms are complicated by employing

harmonics and other features (cf., Guignard & McCauley, 1982) the lack of predictability of the complex stimuli may combine with the increased conflict that differing waveforms may possess over simple ones, and these two elements may explain partly the vastly increased sickness incidence with such stimuli.

We have attempted to relate the three elements in the correlation model of the conflict theory (conflict, salience, and adaptation). Essentially, as correlations get higher, they are likely to be more susceptible to conflict. This is in keeping with correlation theory also. Small differences in correlations at the high (positive and negative) ends of the sampling distribution of correlation coefficients are more noticable (statistically) than differences between correlations which are near zero. High correlations (either positive or negative) are necessary for learning and adaptation because should stimuli not be related one could not profit by continued exposure. An example may illustrate this concept: If a person wears displacing prisms and repeatedly points to a target, receiving feedback from the limb and visually, there is adaptation in the form of decreased error in pointing to an assigned target. Were the person to wear prisms which rotated so that each new feedback input was different from the one before, there would be no adaptation. Therefore, the size of the correlation coefficient is related to the likelihood of adaptation regardless of the sign of the correlation. However, the size of the correlation also implies the magnitude of the disruption because within a sensory channel, the size of the correlation is related to the test/retest reliability of the information delivered over that channel. (N.B. It could either be naturally endowed information processing or it could be acquired.) An operational definition of sensitivity could be related to the ability of the channel to receive accurate information over that channel. In statistical terms this would mean it had high test/retest reliability. We believe that there are areas of sensitivity in bandwidths of sensory channels, and these could be characterized as having higher test/retest reliabilities than in other areas. Stimuli which are not in agreement between two sensory channels, and which have energy in those areas where both channels are sensitive, are likely to yield greater conflict than when in other areas.

It should be mentioned that the conflict can result because of the way in which a sensory channel is naturally endowed to process information, or it could be that it has acquired a new way of responding and the present stimulus is not in accord with what is in the neural store. Thus, the concept that current perceptions are compared to past memories of these perceptions is implied by two studies on simulator sickness where it was noted that more experienced flyers were more likely to become sick in the simulator than pilots with fewer

hours (Miller & Goodson, 1958; McGuinness et al., 1981). However, some conflicts could be immediate without experience (e.g., Coriolis-like stimuli in Slow Rotation Rooms; Kennedy & Graybiel, 1965).

Perceptual conflict is not a new theory. Indeed, as early as 1881, the father of the overstimulation theory (Irwin) wrote, "In the visual vertigo of seasickness there appears to be a discord between the immediate or true visual impressions and a certain habit or visual sense of the fitness and order of things, which passes into consciousness as a distressing feeling of uncertainty, dizziness and nausea," (cited in Reason, 1978, pp. 819-820). Since that time, numerous authors have employed perceptual conflict theory to explain the induction of motion sickness in a variety of situations (e.g., sea, car, space sickness).

Although perceptual conflict theory can often easily explain motion sickness after the fact, Frank et al. (1983), have pointed out three problems with the theory as it now stated.

The first problem with the perceptual conflict theory is that there is presently no good method within the model to determine the magnitude of the conflict for a specific combination of conflicts. Therefore, it is not possible to know if a new, but similar, set of circumstances will lead to a greater or lesser incidence of motion sickness; nor, more importantly, is it possible to identify those factors which are in conflict in existing motion sickness environments such as vehicle simulators.

The second problem is not so much a problem with the theory but with some scientists who apply it. In general, researchers and practitioners have tended to address only conflict between sensory modalities. It is quite possible that conflicts occur within modalities as well as between. Guedry (1970) has suggested, as an explanatory principle for space sickness, that it is possible to have a vestibular/vestibular conflict (canals/otoliths). Analogously, data from Leibowitz and Post (1982) strongly suggest that a visual/visual conflict could also arise, perhaps between the focal and ambient visual systems. Dichgans and Brandt (1973) have reported that increased field of view resulted in increased sickness and that masking the center of the field was without effect. It should be reiterated that the most obvious source of conflict is between visual-vestibular spatial inputs (e.g., reversals), but visual-vestibular temporal discord (e.g., different input delays) also can occur.

Finally, the conflict theory does not explain why sickness does not occur in situations where there is clear-cut conflict

(e.g., tilted rooms) or does occur where little or no conflict is present. [For example, sinusoidal linear oscillation at .20 Hz with vision permitted is generally considered to be very nauseogenic. The slight conflict (a phase advance) which is evidenced in the felt change in direction (Benson, personal communication, 1984) appears disproportionate to the magnitude of the symptomatology.

Despite these problems, there is much to recommend the perceptual conflict theory because it is in accord with so much of the data. For example, the observation that a person suffering from seasickness can reduce the symptoms by positioning himself on deck so that he can see the horizon at sea is used as an argument to support the perceptual conflict theory, since the mismatch between the vestibular and visual systems is less when on deck than when below deck. So too, with less sickness occurring while serving as the operator of a vehicle, instead of as a passeneger. (Although it is recognized in both of these situations that visual information may also better stabilize the head and thus modify the vestibular input.)

Perhaps the two-stage model offered by Sokolov (1963) to explain adaptation of an orienting reaction is a useful way of resolving the discrepancies in perceptual conflict theory. According to Sokolov, an orienting response is analyzed by the cortex, and if coincident with an existing model [cell assembly, (Hebb, 1949); or phase sequence (Sokolov, 1963); or template (Lynn, 1966)], there is no orientation response. Neural stimuli alert the reticular activating system (RAS) and a response results.

Perceptual conflict is invoked for all cases where such conflict occurs, but a frequency resonance principle is sought where a conflict is not apparent. In this manner, a two-process corollary to the perceptual conflict theory may permit a resolution to the noted discrepancies. Each sensory system has a peak sensitivity for a specific bandwidth. These function as filters, much like the RAS in Sokolov's model. Thus, discordant sensory stimuli, impinging upon different sensory systems which are not within the level of maximum sensitivity, may not be a problem. Similarly, two stimuli only a little discordant, but at the peak sensitivity of both systems, could be quite debilitating. Additionally, stimuli at particular combinations of energy, whether discordant or not, are too noisy (or poisonous) to be ignored.

# SECTION VIII

# CONCLUSIONS

## TOWARDS A UNIFIED THEORY

We have reviewed the signs and symptoms, stimuli and response characteristics, anatomical structures, susceptibility factors, and prevalent theories of motion sickness. It is evident from this review that motion sickness is both polysymptomatic and polygenic. It should also be evident from the number of corollaries, principles, postulates, and theories presented, and the examples proposed to explain the outcomes, that "we are light years away from a proper understanding of motion sickness" (Lackner, 1984, personal communication). But we may be closer to predicting its outcome, and perhaps preventing its occurrence. With these provisos in mind, we offer the following:

We would propose that the preceding theories be integrated into one. The theories mentioned above emphasize either the stimulus or response characteristics that lead to motion sickness. However, it appears clear from the literature that the key to understanding motion sickness must include understanding of how the stimulus acts at the receptor level. It is our view that motion sickness is a result of decorrelated sensory channels. This premise, which is in concert with the perceptual conflict theory, states that any stimulus which causes a decorrelation to occur initiates the firing of the chemoreceptor trigger zone (CTZ) and motion sickness. This relationship can be schematized as follows:

Decorrelated Receptors ⟶ CTZ ⟶ Motion Sickness

As with the perceptual conflict theory, correlations between sensory receptors build up over time. Decorrelation occurs when inputs are not in accord with what is expected from the neural store, or with the way in which that system is wired to respond. This causes "troubleshooting" to begin. The toxic reaction, oversimulation and fluid shift theories of motion sickness are all compatible with this notion. Indeed, troubleshooting may be a hypothetical construct for a toxic reaction. Each theory implies that a modification occurs where stimuli are integrated. Overstimulation modifies the receptor through sensitization; fluid shift through pressure changes; poison through varied means.

The autonomic and fear theories of motion sickness also are compatible with the unified theory. The autonomic and fear theories, however, really address responses to motion sickness, not causal factors.

Thus, as Money (1982) contends, Triesman is correct. Presumably, when inimical things happen to the organism, the central nervous system (CNS) interprets these events to mean it has been poisoned. Generally, this interpretation occurs when real poisons are administered, but in those special cases where altered and rearranged perceptions occur, if the vestibular system is implicated, the system interprets that the organism has been poisoned. Under some conditions, the body possesses resonances which, in the case of .20 Hz or so, the system also interprets as being poisoned. [It is wondered whether .20 Hz, or another resonance, would have an adverse (i.e., it's poison!) effect with visual stimuli alone. If so, such a finding would have strong heuristic value for simulator sickness.]

The above conceptualization eliminates problems associated with the perceptual conflict theory as follows: 1) sickness without conflict and conflict without sickness - resonance at .20 Hz is just like poison; 2) no measure of conflict - each modality has channels and peak sensitivities, and the conflict occurs when spatial (gain) and temporal (phase) aspects of the stimulus are not in accord. If the lack of accord occurs at places where the two channels are both sensitive, there is more disruption (poisoning) than at places where one or the other may be insensitive. Presumably, if discord occurs where both sensory modalities are insensitive, little poisoning will be perceived. Guedry (1968) is correct in asserting that there is a conflict within the vestibular system during head movements in space flight, and it is equally possible that there may be conflicts between two visual systems (Leibowitz & Post, 1982) during, for example, perceived forward motion in a flight simulator.

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